unlikely to be of value and often causes unnecessary anxiety. Nutrition is better assessed using skinfold calipers (which are also cheaper and more portable than weighing scales) to measure directly the thickness of subcutaneous fat.[1]

Accurate height measurement (supine length in infants under 2 years) is a sensitive guide to child health.[2] Growth velocity (calculated from repeated measurements of height at intervals) represents the current dynamics of growth much better than a single measurement, which reflects previous growth. Regular, accurate measurement of children can identify those who would benefit from medical, social, or educational intervention.[3]

Many height measurements in hospital and the community are inaccurate and misleading because of careless techniques and inadequate apparatus. Suitably accurate, cheap, and portable apparatus is now widely available for use in primary care, and measuring techniques eliminating postural drops and positional errors are readily learnt by motivated staff. Supine length in children under 2 years can generally be measured accurately with the help of an assistant.

Collected accurate growth (height) data in children have important benefits beyond those to the individual—as an index of the health of a population or a subgroup (for example, ethnic group or social class). British data are not available and would be valuable.

Many who care for children lack the skill to measure them accurately, plot measurements on a growth chart, and interpret the data obtained. As the report states, such understanding is essential for growth monitoring. More must be done to make those who look after children aware of the need to measure height accurately and regularly throughout childhood and to train them to do so.

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1 Polnay L. Child health surveillance. *Br Med J* 1989;299:1351-2. (2 December.)
2 Hall DMB, ed. *Health for all children, the report of the joint working party on child health surveillance*. Oxford: Oxford University Press, 1989.
3 Tanner JM, Whitehouse RH. Revised standards for triceps and subscapular standards in British children. *Arch Dis Child* 1975;50:142-5.
4 Tanner JM. *Fetus into man*. 2nd ed. Ware: Castlemead Publications, 1989.

SIR,—*Health For All Children*, discussed by Dr Leon Polnay[1] and Dr D M B Hall,[2] is the result of a working party set up by groups representing paediatricians and general practitioners, neither of whom is disinterested. The British Paediatric Association suggested some years ago that senior clinical medical officers in the child health service should be replaced by "community paediatricians" who would work part time as paediatricians in the hospital and would take part in the on call duty roster. Similarly, much of the interest in taking over child health surveillance by general practitioners has been tied to the proposal that extra payments would be made for such a service. Practitioners who have a real interest in this work provide such a service already for patients on their lists. Bodies that actually represent the medical officers who work in the child health service were not invited to join the working party.

Child health surveillance requires a different outlook from clinical medicine, and it is not easy for clinicians whose whole training has been directed to the diagnosis and treatment of disease to stop thinking in such terms and abandon their prescription pads. Clinicians are not the most appropriate group to advise on a child health service that they do not fully understand.

Developmental assessment and child health surveillance were pioneered by the former child health group of the Society of Medical Officers of Health which started running full time training courses of six weeks' duration for doctors some 30 years ago. In the early 1970s when the Faculty of Community Medicine was formed community health doctors were not eligible for membership. Fortunately, a number of medical schools started to run training courses in child development to fill the need that resulted. There was, however, no organisation or body monitoring the standard or content of those courses, which varied widely.

Following the formation of the Faculty of Community Medicine residual members of the Society of Community Medicine sought to promote the interests of community health as well as community medicine. In 1988 the society (which has since changed its name to the Society of Public Health) was instrumental in establishing a new Faculty of Community Health to produce syllabuses, set standards, and appoint examiners. In future, membership of the Faculty of Community Health should be evidence of eligibility for posts as senior clinical medical officer or as consultant in community child health—more appropriate to the needs of the clients and of the child health and education services than "community paediatricians."

We hope that this faculty will provide training for general practitioners in child health surveillance and that appropriate diplomas will be established.

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1 Polnay L. Child health surveillance. *Br Med J* 1989;299:1351-2. (2 December.)
2 Hall DMB. Child health surveillance. *Br Med J* 1989;299:1352-3. (2 December.)

Lee, P.N.

Passive smoking and cardiorespiratory health in Scotland

SIR,—In an earlier letter[1] I claimed that misclassification of active smoking state can explain the fact that Mr David J Hole and his colleagues[2] found weak positive associations between passive smoking and a number of indicators of cardiorespiratory health in the Scottish prospective study. In their reply Mr Hole and colleagues presented calculations to justify their view that the effect of misclassification is to produce "only small biases in the relative risk for passive smokers," with the reported relative risk "well in excess" of that produced by this form of bias.[3] Unfortunately, these calculations are grossly in error and therefore highly misleading.

The error lies in basing calculations on results for men and women combined without adjustment for sex. Table I of the original paper[2] shows a clear association between the smoking habits of the index case and the cohabitee, with the concordance (cross product) ratio being 2·32 for men and 2·19 for women. An appropriate estimate of the concordance ratio for the sexes combined with sex adjustment by the Mantel Haenszel procedure[4] is 2·25. If, inappropriately, the concordance ratio is calculated from the pooled data, a much lower figure of 1·29 is obtained, and this masks most of the true association. This is important because it can readily be shown that the concordance ratio provides the upper limit to the extent of the observed relative risk from passive smoking due to misclassification of smoking habit (assuming a true relative risk of 1·0). Table I shows that when correctly calculated the observed relative risk can far exceed the value of 1·20 stated by Mr Hole and his colleagues to be "the largest risk to be among passive smokers due to this form of bias."[3]

TABLE I—*"Observed" relative risks for passive smoking for varying denial rates of smoking**

Rate of denial (%)	Relative risks for passive smoking			
			Sexes combined	
	Men	Women	Adjusted†	Unadjusted‡
2	1·74	1·25	1·40	1·12
4	1·95	1·42	1·58	1·18
6	2·06	1·54	1·70	1·20
8	2·11	1·63	1·78	1·22
10	2·15	1·70	1·84	1·23

* Assuming "true" relative risks of 1·0 for passive smoking and 20·0 for active smoking.
†Adjusted for sex using weights $N_1N_2/(N_1+N_2)$, where N_1 and N_2 are the observed numbers of exposed and unexposed subjects. This is a conservative approximation to the true adjusted figure, which cannot be calculated precisely from the data provided by Hole *et al*.
‡As given by Hole *et al*.[2]

The question arises as to the extent that this source of bias can explain all the reported relative risks for active and passive smoking seen in the Scottish study. Table II gives some insight into this question, showing "observed" and "true" relative risks assuming a 4% denial of smoking, a figure consistent with data from many studies of the issue.[4] Comparing the "observed" relative risks of active and passive smoking with those given in table VII of the original paper[2] shows that there is no problem whatsoever in reconciling the data with the bias hypothesis for most of the cardiorespiratory endpoints. For example, relative risks of 3·77 (active) and 1·21 (passive) for hypersecretion are both very close to the values given in table II for a "true" active risk of 5 (1·23 and 3·63, respectively).

TABLE II—*"Observed" relative risks for passive and active smoking for varying "true" relative risks for active smoking**

"True" relative risks		"Observed" relative risks	
Passive smokers	Active smokers	Passive smokers	Active smokers
1	30	1·70	9·17
1	20	1·58	7·88
1	10	1·39	5·62
1	5	1·23	3·63
1	3	1·13	2·50
1	2	1·07	1·81

* Assuming 4% of smokers deny smoking. Results are for sexes combined adjusted for sex as in table I.

Only two endpoints deserve special comment. The first is death from lung cancer, for which risks of 10·64 (active) and 2·41 (passive) were observed. The confidence interval for the risk with passive smoking was enormously wide (0·45 to 12·83), and the point estimate of risk was higher than that in any of over 20 other, larger, studies on the issue.[5] I have claimed elsewhere that misclassification of active smoking state can explain the average relative risk for passive smoking of about 1·3-1·5 seen in epidemiological studies.[5] I retain this view but have never stated that it explained the figure in the Scottish study, of 2·41, to which chance has clearly contributed substantially.

The other endpoint is ischaemic heart disease, for which risks of 2·27 (active) and 2·01 (passive) were observed. Although the risk with passive smoking is significant (95% confidence interval 1·21 to 3·35) and the lower confidence limit is slightly above the bias expected, I do not find this convincing evidence of a true effect of passive smoking. This is partly because the significance level is not high, bearing in mind the number of endpoints studied, and partly because the point estimate of relative risk for passive smoking is difficult to reconcile with that for active smoking, bearing in mind that smokers have much higher active and passive exposure to the constituents of smoke, in the form of both mainstream and sidestream smoke, than do passively exposed non-smokers. More evidence is clearly needed here. The American Cancer Society million person study

accumulated 153 deaths from lung cancer and many thousands of deaths from ischaemic heart disease in non-smokers. The effect of passive smoking on lung cancer has been looked into.[1] It is a pity that its effect on ischaemic heart disease has not.

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1 Lee PN. Passive smoking and cardiorespiratory health in Scotland. *Br Med J* 1989;299:742. (16 September.)
2 Hole DJ, Gillis CR, Chopra C, Hawthorne VM. Passive smoking and cardiorespiratory health in a general population in the west of Scotland. *Br Med J* 1989;299:423-7. (12 August.)
3 Hole DJ, Gillis CR, Chopra C, Hawthorne VM. Passive smoking and cardiorespiratory health in Scotland. *Br Med J* 1989;299:1100-1.
4 Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. *J Natl Cancer Inst* 1959;22:719-48.
5 Lee PN. Passive smoking and lung cancer: fact or fiction? In: Bieva CJ, Courtois Y, Govaerts M, eds. *Prevention future of indoor air quality*. Amsterdam: Elsevier, 1989:119-28.
6 Garfinkel L. Time trends in lung cancer mortality among non-smokers and a note on passive smoking. *J Natl Cancer Inst* 1981;66:1061-6.

AUTHORS' REPLY.—Our calculations are neither incorrect nor misleading. Mr Lee is attempting to show how large a bias can be introduced into estimates of relative risk for passive smokers due to active smokers misclassifying themselves as non-smokers. In doing so he has produced biases that are excessive because we can show his assumptions are false. His main mistake has been to assume that the "true" relative risk for lung cancer is the same for male and female smokers (his table I). Also, although the extent of smoking denial for our study is not known, we can put an upper boundary on it.

Our original study estimated the relative risk of lung cancer among active smokers as 8·49 for men and 3·33 for women.[1] Table I shows, under Mr Lee's assumptions, that "observed" relative risks for active smokers would be larger for women than [illegible] n. This is incompatible not only with what we [illegible]ve observed but also with all other reports we know of. Thus his assumption that the same "true" relative risk holds for both men and women is untenable. Also, if we accept Mr Lee's theoretical range of possibilities for the rates of denial of cigarette smoking then the outcomes become even more unlikely. For each rate of denial of 4% and over suggested by Mr Lee the relative risk for male active smokers is progressively well below that observed in our study (table I). Above a denial rate of 8% the "observed" relative risk for male passive smokers exceeds that for active smokers. Our data are, however, compatible with denial rates of up to 2% and a "true" relative risk of 4 for female smokers.

Mr Lee questions the extent to which misclassification can explain all the reported relative risks for active and passive smoking seen in our study. Table II shows the relative risks for active smokers found in our study for each endpoint and the "true" relative risks with which these are compatible, assuming a rate of denial of smoking of 2%. For example, the relative risks for all causes of death associated with active smoking are 1·85 for men and 1·87 for women. These figures are compatible with a "true" relative risk of 2, given a denial rate of 2%. The figure of 5 that Mr Lee quotes in his letter may be appropriate for some of the endpoints used but certainly not for all.

The final two columns of table II show the passive smoking relative risks found in our study for each of the endpoints compared with those that could have occurred through the type of bias Mr Lee attributes to our study. In particular, the differences are quite noticeable for the four categories of mortality. Thus misclassification can bias estimates of relative risk for passive smokers that use assumptions compatible with our estimates for active smokers. The size of these biases does not, however, explain our passive smoking results.

What is striking about our results is their consistency across a wide range of endpoints in addition to lung cancer and especially for ischaemic heart disease. This is supported by our findings of a dose-response relation for each of these. Even though Mr Lee reaffirms his view that misclassificaton of active smoking state can explain the average risk of lung cancer with passive smoking, we welcome his implication that the effect of passive smoking on ischaemic heart disease is worth further investigation.

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TABLE I—*"Observed" relative risks for active and passive smokers for varying denial rates of smoking**

Rate of denial (%)	Relative risks for active smoking		Relative risks for passive smoking	
	Men	Women	Men	Women
1	10·34	16·20	1·53	1·15
2	6·90	13·42	1·74	1·25
3	5·14	11·53	1·57	1·34
4	4·10	9·99	1·95	1·42
6	2·88	7·89	2·06	1·54
8	2·20	6·48	2·11	1·63
10	1·76	5·45	2·15	1·70

*Assuming "true" relative risks of 1·0 for passive smoking and 20 for active smoking.

TABLE II—*Relative risks found in study compared with "true" relative risks for active smokers and "observed" relative risks for passive smokers**

Endpoint	Active smokers				Passive smokers	
	Men		Women		Both sexes	
	Study finding	"True" relative risk	Study finding	"True" relative risk	Study finding	"Observed" relative risk
Infected phlegm	4·03	6·0	3·82	5·0	1·34	1·14
Persistent phlegm	4·23	6·0	3·93	5·0	1·19	1·14
Dyspnoea	1·65	1·9	1·37	1·4	1·09	1·03
Hypersecretion	2·95	5·0	4·15	5·0	1·21	1·13
Angina	2·13	2·7	1·44	1·5	1·11	1·05
Major abnormality in electrocardiogram	1·57	1·8	0·92	1·1	1·27	1·02
All causes of death	1·85	2·0	1·87	2·0	1·27	1·04
Ischaemic heart disease	1·36	3·0	2·89	3·0	2·01	1·07
Lung cancer	8·49	20·0	3·33	4·0	2·41	1·26
All causes of death related to smoking	1·90	3·0	2·45	3·0	1·30	1·07

*Assuming 2% of smokers deny smoking. The results for both sexes combined have been adjusted for sex using weights $N_1N_2/(N_1/N_2)$, where N_1 and N_2 are observed numbers of exposed and unexposed subjects.

Congenital malformations

SIR,—In her editorial on congenital malformations Professor Eva Alberman comments on the excess rate of deaths from malformations, particularly neural tube defects, in infants of mothers born in Pakistan.[1] In the studies referred to only perinatal deaths were considered. Many neural tube defects in this country are now detected by prenatal screening programmes, and women may opt for termination of the pregnancy when found to have an affected fetus,[2] so these studies may not reflect the true incidence of neural tube defects. Asian women tend to book later for their antenatal care,[3] and this may account for the high contribution of neural tube defects to perinatal mortality: second trimester screening would be available to a relatively smaller proportion of Asian women. Furthermore, they may find termination of pregnancy unacceptable on religious grounds.[4] We have investigated the overall incidence of neural tube defects by ascertaining all those affected fetuses detected by prenatal screening with ultrasonography, as well as all those found in the perinatal period. We have also tried to determine factors that may be important in explaining any racial differences in the incidence.

We reviewed the maternity ultrasonography department records, neonatal and labour registers, and necropsy reports from January 1980 until the end of December 1987 in one district general hospital to ascertain all fetuses, stillbirths, and neonates with a neural tube defect. The maternal notes were then inspected to determine the date of booking for antenatal care, if and when an ultrasound scan was performed, and whether a termination of pregnancy was offered.

In the Pakistani population there were 11 neural tube defects in a total of 3777 births (2·91 per 1000); there were 32 neural tube defects in 28 834 births to white women (1·11 per 1000) (table).

Incidence of neural tube defects in fetuses and babies of white and Pakistani women, 1980-7

	White women	Pakistani women
Detected by routine ultrasound scan	17	5
Pregnancy terminated	17	4
Pregnancy continued		1
Not detected by routine scan	15	6
Scan not available	12	3
Not detected by scan	2	1
Booked too late for scan	1	1
Did not attend for scan		1
Total neural tube defects	32	11
Total births	28 834	3 777
Incidence per 1000 births	1·11	2·91

Routine examination with ultrasound was introduced only in 1984 and hence was not available to many of the women included in this study. The incidence of neural tube defects in the Pakistani population was significantly higher than that in the white population ($p=0·013$, Fisher's exact two tailed test; relative risk 2·62, 95% confidence interval 1·19 to 5·34). One woman in each group booked too late for routine prenatal screening, and one Pakistani woman failed to attend for the scan. These numbers are small, but it is of note that the mean gestation at which these women booked was 18·2 weeks in the Pakistani group as compared with 14·3 weeks in the white group.

Six of the 11 Asian babies with neural tube defects were born to women with a consanguineous marriage.

We have shown that there is a real increased incidence of neural tube defects in the Pakistani population, with late booking and reluctance to terminate an affected pregnancy contributing minimally to the increased incidence found in perinatal deaths. Changes in customs are difficult to encourage but may well occur spontaneously as

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Incidence of neural tube defects in fetuses and babies of white and Pakistani women, 1980-7

	White women		Pakistani women	
Detected by routine ultrasound scan		12		5
Pregnancy terminated	17		4	
Pregnancy continued			1	
Not detected by routine scan		15		6
Scan not available	12		3	
Not detected by scan	2		1	
Booked too late for scan	1		1	
Did not attend for scan			1	
Total neural tube defects		32		11
Total births	28 834		3777	
Incidence per 1000 births	1·11		2·91	

Routine examination with ultrasound was introduced only in 1984 and hence was not available to many of the women included in this study. The incidence of neural tube defects in the Pakistani population was significantly higher than that in the white population ($p=0·013$, Fisher's exact two tailed test; relative risk 2·62, 95% confidence interval 1·19 to 5·34). One woman in each group booked too late for routine prenatal screening, and one Pakistani woman failed to attend for the scan. These numbers are small but it is of note that the mean gestation at which these women booked was 18·2 weeks in the Pakistani group as compared with 14·3 weeks in the white group.

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